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The Mosaic Disease of Sweet Potatoes With Special Reference to Its Transmissibility

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SUMMARY

Sweet potato mosaic has been reported from Arkansas, Florida, Mississippi, Texas, and Kansas. Observations over a period of six years in Arkansas indicate that the disease, though well scattered, is not prevalent on any large percentage of plants. Up to the present only the Nancy Hall variety has been found susceptible.

Artificial infection experiments conducted over a period of years and involving large numbers of plants show that the disease is infectious. Infections have been obtained in two ways, by rubbing leaves of healthy plants with macerated foliage of diseased ones and by injecting juice of diseased plants into the stems of healthy ones.

The largest percentage of infections obtained, somewhat over 20 per cent, resulted from treating healthy plants in two ways, by rubbing diseased material on the leaves and, in addition, injecting juice from mosaic plants into the stem.

No infections have been obtained in the period covered by one growing season or by three successive growing seasons in which the fleshy roots were used for propagation after the lapse of the intervening winter seasons.

The only method by which the disease has been transmitted thus far is by the continuous growth of inoculated plants over a period of 12 to 13 months. This consists of inoculating plants in the field early in the growing season and giving opportunity for good growth throughout the season, growing cuttings from such plants over winter in the greenhouse, followed by transplanting into the field the following year.

Observations of plants artificially infected have revealed the following. The disease develops gradually, becoming more severe as time advances, until a certain maximum is reached. Frequently only a small proportion of the runners are reported as showing the disease on any one plant. There is also noted a recedence of the disease on some plants which at one time were severely infected.

Several explanations of the foregoing observations are presented with the suggestion that the disease in its various manifestations is due to one causal agent instead of several as in certain combinations of "degeneration diseases" of Irish potatoes.

The work herein reported suggests that the disease is not capable of being transmitted to healthy plants under natural conditions in the northern sweet potato sections, while in southern-most regions, as around the Gulf of Mexico, there is ample opportunity for transmission.

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SWEET POTATO MOSAIC

WITH SPECIAL REFERENCE TO ITS TRANSMISSIBILITY

COCKAYNE BOSTON

The Mosaic Disease of Sweet Potatoes With Special Reference to Its Transmissibility

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Ever since sweet potato mosaic has been known, its ability to infect healthy plants has been in doubt. Indeed some investigators have questioned the use of the term "mosaic" for this disease because they were unable to prove that it possessed any infectious properties *(22)*. The aim of this bulletin is to present evidence which has accumulated in the course of about six years, showing, without any doubt, that the disease is transmissible and infectious. Furthermore, it will be shown that the length of time involved in the period of incubation is so unique as to make this disease of special interest.

It may be worth while to review briefly the known facts concerning sweet potato mosaic. It was first discovered in 1919 in Arkansas *(15)* and has subsequently been reported by other investigators from four additional states, ranging from Florida to Kansas *(2)*. Only one variety, Nancy Hall, which happens to be one of the more important commercial varieties grown in the South, has until the present time been found susceptible. It should be noted, however, that the difficulties which had to be overcome in proving infectivity of the disease have considerably retarded work on varietal susceptibility. Also, the possibility that other convolvulaceous species may be susceptible needs to be borne in mind.

As to symptoms, these have already been fully discussed *(15)* and may be briefly summarized as follows:

(1) A very conspicuous variegated or mottled effect on foliage in the form of very small, light-colored dots, streaks and blotches thickly or sparsely scattered over the leaf-blades (figs. 1 [colored] and 2); (2) instead of small, light-colored areas, larger irregular blotches of yellow-green or gray-green interspersed between the normal appearing dark- or blue-green color are often to be seen (fig. 1 [colored]); (3) extremely irregular margins of blades in severe cases, but not so uneven in lighter infections (fig. 1 [colored]); (4) very noticeable shortening of internodes in heavy infections, resulting in a more upright bushy appearance and conversely a lack of spreading or running habit; (5) a wrinkling, curling, and pouchiness of portions of leaves or of whole leaves; and (6) a greater or less amount of reduction in foliar surface, depending upon the severity of infection. In severe cases, this reduction is so great that very few or no marketable roots are produced.

Losses occasioned by this disease have not been large. While severely infected plants, as noted above, are almost com-

plete failures, such plants have not been found in any great abundance. While field inspectors have reported considerable loss in some particular field, the disease, though well scattered, has not been found on large numbers of plants. As this malady belongs to a group which as a whole is widespread and causes great damage, notably Irish potato mosaics, tobacco mosaic, sugar-cane mosaic, etc., it may be asked, why is the sweet potato mosaic so limited in its distribution? The answer is not certain but there are two possibilities, first, that the disease is of recent origin in the United States, having been recently brought in, or, possibly, having spread recently from some other susceptible host, and second, that the length of time involved in disease production is so much greater than in other comparable diseases, as will be presently shown, that its spread has been considerably retarded.

There is another malady occasionally observed on several varieties of sweet potatoes which may be mistaken for the mosaic. This takes the form of large whitish or creamy-yellow blotches, or streaks, one blotch frequently involving a large part or the whole of the leaf blade (see fig. 3). It never causes any marginal irregularities, or any curlings of blades, and it never causes reduction in size of internodes. It rarely involves more than a few leaves on any one plant. In gross symptoms it is comparable to a number of chlorotic disturbances, non-infectious, which have been described in various types of plants, as for example, the variegated sycamore maple, and variegated box elder, illustrated by Clinton (*5* pl. 25), or the chlorotic streaked and blotched Indian corn, illustrated by Lindstrom *(11)*.

INFECTION EXPERIMENTS

As reported in 1920 *(15)* all efforts at artificial infections resulted in apparent failure. That is, as far as outward symptoms were concerned, all inoculated plants remained entirely free from any signs of disease. The inoculum was applied to young as well as to old plants and the applications consisted of badly diseased foliar material, rubbed into young and old leaf blades, leaf petioles and stems of plants; also, hypodermic injections into stems, petioles and midribs; heavy applications of expressed juice from diseased leaves into various sized cavities of fleshy roots, such roots being used for propagation; tying halves of diseased roots with healthy roots and using slips from the healthy halves; grafting healthy shoots on diseased plants; all such applications failed to produce any recognizable symptoms on the healthy parts.

The work previously reported has been repeated for a number of years and on a rather large scale. Altogether some 500 inoculations of various types have been attempted and without exception not a sign of disease has been obtained during the

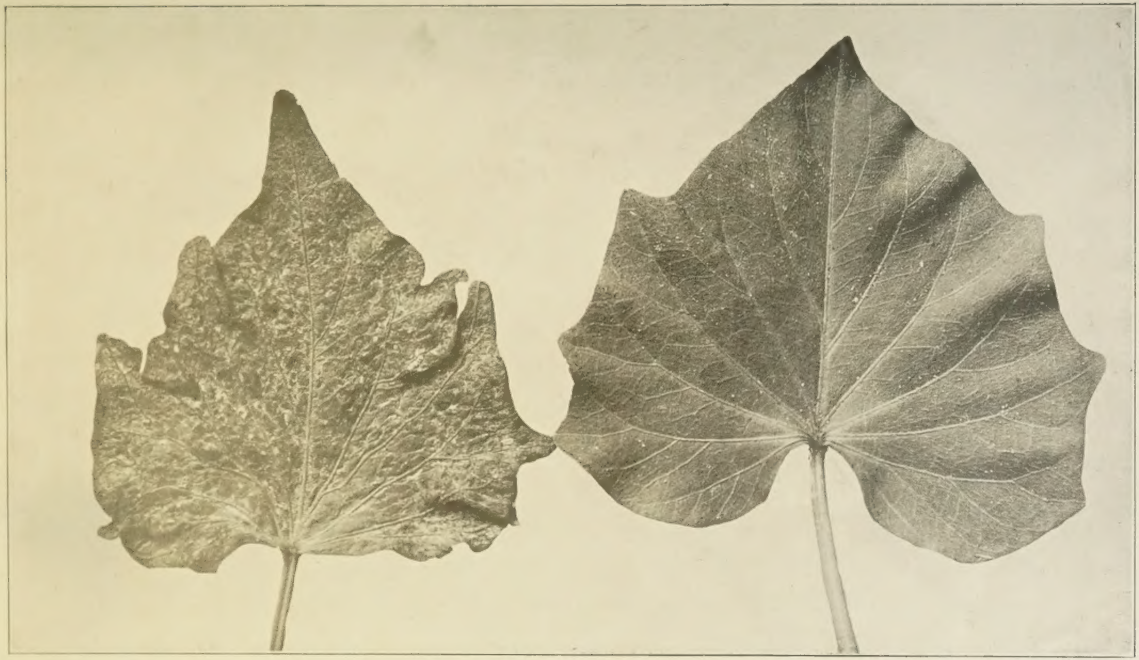

FIGURE 2. Left-hand leaf typifying a common form of sweet po tato mosaic; the chlorotic areas are minute spots. Right-hand leaf represents foliage from a healthy plant (Nancy Hall) growing close to a diseased one.

current season in which the inoculations have been made. Other investigators including Weber in Florida *(21)*, McClintock in Tennessee (in conversation with the writer), and Weimer *(22)* in Washington, D. C., have all tried and all report failure in transmitting the disease to healthy plants. Indeed, Weimer's failure to obtain infections has led him to question the use of the term "mosaic" for this disease. Six years ago the present writer also queried as follows (*15* p. 4): "The question naturally arises as to the advisability of using the name 'mosaic' for this disease" for "investigations so far have failed to show the possession of any infectious properties." Happily the following statement was added to the foregoing: "On this point, however, the writer feels that the methods used thus far to establish this feature may have been at fault and that by using other means the disease may yet be found to be infectious."

Beginning with the work of Bauer in 1906 *(4)* it has been known that certain plants may possess an infective principle, i. e., may act as disease carriers, and yet show no recognizable symptoms of infectious chlorosis themselves. His findings have been substantiated and further extended among the true mosaic diseases by the work of Allard *(1)*, Nishimura *(14)*, Melhus *(12)*, Murphy and McKay *(13)*, Atanasoff *(3)*, Elmer *(8)*, and others. In other words, it is known that some plants, while harboring the infective principle, either lose the mottling symptoms or at no time show any outward signs of disease. (Johnson *(10)* has recently shown that under certain environmental conditions at least some of these "masked carriers" may show disease symptoms.) Of course only the work of one or two investigators had appeared up to the time of the writer's first publication, in 1920, but that it was clearly in his mind may be seen from the following quotation (*15* p. 6): "It is not impossible to conceive of a situation where a plant may be infected without showing the disease and that proof of infection is not obtainable until the roots of such plants are used for seed purposes." Attention is called to this because if it had not been for that attitude the current year's work on transmission experiments would not have been carried forward, as will shortly be discussed. As Schultz and his associates *(20)* had noted that Irish potato plants infected with mosaic late in the growing season may not show disease symptoms until the following year, it seemed desirable to continue the growth of inoculated plants at the end of the natural growing season.

To test the foregoing idea three cuttings were made from one of the plants which had been inoculated with disease-carrying juice by means of a hypodermic injection in July, 1919. The cuttings were made shortly before frost in October. These were set out in large sized pots in a greenhouse and kept under close observation. Not a sign of disease appeared up to May, 1920,

FIGURE 3. A non-infections chlorosis of sweet potatoes, variety Triumph, which may be confused with mosaic. The chlorotic areas are usually extensive but aside from this the plants appear normal.

when they were transplanted in the field. They were now of good size, with a few fleshy roots noticeable as they were being transplanted. Up to July, 1920, no mosaic symptoms appeared on these or any of the plants inoculated in the current year. In order to show the scale of the various tests, the following table is presented:

FIELD TESTS OF 1920

Plot Number	Number of plants	Types of Treatment
(1)	3	Representing cuttings from vine injected in 1919
(2)	3	Representing slips from ½ healthy root tied to ½ mosaic root[1]
(3)	8	Representing slips from mosaic root[2]
(4)	10	Representing slips from root treated with 2 c. c. of $(NH_4)_2SO_4$[3]
(5)	5	Representing slips from root treated with 1 c. c. of $(NH_4)_2SO_4$[3]
(6)	8	Representing slips from root treated with ½ c. c. of $(NH_4)_2SO_4$[3]
(7)	6	Representing slips from root treated with 1 c. c. mosaic juice[4]
(8)	6	Representing slips from root treated with 1 c. c. mosiac juice[4]
(9)	2	Representing slips from root treated with 5 c. c. of $(NH_4)_2SO_4$
(10)	10	Representing slips from root treated with water
(11)	9	Representing slips from root treated with 2 c. c. mosaic juice[4]
(12)	9	Representing slips from root treated with 5 c. c. of $(NH_4)_2SO_4$
(13)	6	Representing slips from root treated with 1.5 c. c. of $(NH_4)_2SO_4$
(14)	3	Representing healthy slips whose stems were injected with mosaic juice[5]
(15)	3	Representing healthy slips whose stems were injected with mosaic juice[5]
(16)	2	Representing healthy slips whose leaves had been rubbed with mosaic leaves
(17)	4	Representing healthy slips derived from same root as (15) and (16)
(18)	4	Representing root treated with 4 c. c. of $(NH_4)_2SO_4$
(19)	5	Representing slips from root treated with 2 c. c. of $(NH_4)_2SO_4$
(20)	3	Representing slips from root treated with 0.25 c. c. $(NH_4)_2SO_4$
(21)	6	Representing slips from root treated with 0.5 c. c. water
(22)	3	Representing slips from root treated with 1.0 c. c. water
(23)	5	Representing slips from root treated with 0.5 c. c. mosaic juice
(24)	5	Representing slips from root treated with 1.5 c. c. $(NH_4)_2SO_4$
(25)	5	Representing slips from root treated with 1.0 c. c. $(NH_4)_2SO_4$
(26)	5	Representing slips from root treated with 2.0 c. c. $(NH_4)_2SO_4$
(27)	3	Representing slips from root treated with mosaic juice[6]
(28)	2	Representing slips from root treated with $(NH_4)_2SO_4$
(29)	4	Representing slips from root treated with $(NH_4)_2SO_4$
(30)	1	Representing slip rubbed with mosaic leaf while attached to root
(31)	2	Representing slips from root that grew No. (30)
(32)	1	Representing slip rubbed with mosaic leaf while attached to root
(33)	4	Representing slips from root injected with 5 c. c. mosaic juice[4]
(34)	2	Representing slips from root derived from mosaic mother plant
(35)	5	Representing slips from root derived from mosaic mother plant
(36)	7	Representing slips from root injected with water
(37)	7	Representing slips from root injected with 2.5 c. c. $(NH_4)_2SO_4$
(38)	4	Representing slips from root injected with 2.0 c. c. $(NH_4)_2SO_4$

[1]Root halves had not united when dug up.

[2]Mother plant with a severe case of mosaic in 1919.

[3].002 per cent of $(NH_4)_2SO_4$ had been applied in wells made in the fleshy roots with the idea that chlorotic symptoms might be produced on the slips.

[4]The juice representing macerated, diseased leaves, had been applied in wells; lids, representing the outer portions of the tissue cubes which had previously been removed, were then put on and to insure against possible desiccation and drainage, melted paraffin was applied around the edges.

[5]The juice had been injected while the slips were still attached to the old, mother root, prior to their being transplanted. After the injection, accomplished by hypodermic needle, the wound had been covered with melted paraffin.

[6]The juice in this case was injected after the shoots had pushed out. In contrast to the application prior to the formation of shoots, it was felt that application to the root after the shoots had appeared might assist in disease development conceivably through a more rapid translocation.

FIGURE 2.—Shoot at the right showing typical mosaic symptoms, consisting of yellow areas, puckerings, and marginal distortions. Compare with other shoots from the same mother plant. This plant represents a healthy one which received a hypodermic injection of mosaic juice in 1919. No signs of disease appeared that year. Cuttings were made of this plant in the fall of 1919. These were grown over winter in the greenhouse without showing any disease. The following May, 1920, the greenhouse plants were set out in the field and in July the mosaic shoot, shown at the right, appeared.

All the plantings were made in May. After the plants had become established and were growing vigorously various inoculations were attempted other than those in the foregoing list. On July 17 two runners coming from healthy plants were grafted onto two diseased plants. One of these runners failed to grow, the other made proper contact and grew considerably before frost. On July 23 crushed mosaic leaves were rubbed vigorously into leaves of three plants in plot (36). Aside from plants in plot (1) which will be discussed below, only the plants obtained from roots derived from diseased mother plants in plots (3), (34), and (35) showed mosaic symptoms. As to the plants in plot (1), representing hypodermic injections made the previous season, one of the plants on July 22 was noted to possess a single shoot with every leaf showing unmistakable symptoms of the disease (see fig. 4), and on Aug. 8, two shoots were noted on another plant of the same series showing unmistakable symptoms. Up to the end of the growing period no other signs of mosaic were noted. Out of some 162 plants, not derived from previously diseased plants, a large number representing such diverse means of inoculation as root grafting, root injections, leaf applications, and stem grafting, not a single case of mosaic was discerned. It should also be noted that mosaic plants were growing alongside of healthy plants, the runners in quite a few cases overlapping. As no infections had been obtained in the previous year's work there seemed to be no good reason for separating the plants, and again no infections were obtained.

As to the three plants in plot (1), representing hypodermic injections the previous season as well as continuation of growth in the intervening winter in the greenhouse, two of these plants as already noted put out shoots with clear mosaic symptoms. The third plant, however, failed to show any signs of disease and as will be brought out later this occurrence of diseased plants and healthy ones from the same mother plant is one of the interesting features of this disease. Not only was one of the plants entirely free from the disease in this series, but of the other two plants, the healthy shoots which had grown out prior to the appearance of the mosaic shoots, remained healthy in appearance in spite of the comparatively large amount of growth which they made subsequent to the mosaic development. Final and complete proof of transmission in these two plants was obtained when their fleshy roots were used for propagation. The shoots from seven of such roots when propagated in the greenhouse (April, 1921) all showed the typical mosaic symptoms, and when slips derived from them were transplanted in the field, the resulting plants all developed typical mosaic.

The above series, however, was not considered conclusive for two reasons, first, the relatively small number of plants in-

oculated by means of rubbing mosaic foliage on healthy leaves, and second, the small number of plants in which transmission was obtained.

In 1921 and in 1922 additional series of field tests were undertaken with the various types of inoculations attempted in 1920, presented in the foregoing table. Slips were grown in the greenhouse from the 1920 and 1921 roots, respectively, and transplanted in the field about the middle of May. Altogether some 200 hills were utilized each year. In no case was there any sign of disease transmission other than those noted in 1920, in spite of the fact that three successive growing seasons had intervened since the original inoculations. Of course the numbers of hills representing inoculations by means of hypodermic injections or even by means of rubbing diseased material on healthy foliage were relatively few. On the other hand over 100 hills each year represented inoculations made into roots and for three years healthy plants had been grown alongside of diseased plants, methods sufficing to insure infections in most if not all the other mosaic diseases known. Could this mean that the transmission obtained in the two hills of 1920 were doubtful or could it be explained on the basis of the different methods utilized? In the two transmission cases, as already explained, cuttings were made in the inoculated plant and grown over winter in the greenhouse, while in all the other work the fleshy roots had been stored over winter and then utilized the following spring for the growing of slips.

Obviously the proof of transmissibility was still inconclusive. In 1924 another series of inoculations was made as follows:

Plot Number	Number of plants	Types of Treatment
(1)	6	Badly mosaic plants from formerly diseased material
(2)	7	Rubbed vigorously 5 leaves of each plant with macerated, diseased foliage[1]
(3)	13	Same treatment as in plot (2)
(4)	11	Injected by means of hypodermic needle with juice from diseased plants[2]
(5)	9	Injected with hypodermic needle as well as rubbing diseased material on several leaves of each plant
(6)	9	Untreated

[1]Plants grown in the same row with the mosaic ones.
[2]Juice obtained by macerating mosaic leaves in a mortar and straining through a coarse cheese cloth.

The plants were set out in May and variously treated on June 17, after they had become well established. The results of these tests were: first, the plants from formerly diseased ones all developed severe cases of mosaic; second, no disease

appeared on any of the other plants, uninoculated or inoculated up to late fall, a growth period of approximately four months after inoculation.

Toward the end of October, shortly before frost, three cuttings were taken from each plant and set out in eight-inch pots in the greenhouse. Aside from the original diseased ones, 147 plants representing checks as well as inoculated, were grown throughout the winter of 1924-1925. Again no signs of disease developed. In May, 1925, each potted plant including the soil around the roots was set out in the field.

About the middle of July, three plants of plot (5) (1924 injections) showed clear mosaic symptoms, one plant of plot (2), and one of plot (3), a total of five plants out of about 60 inoculated ones. (One-half of the plants grown in the greenhouse were discarded for various reasons.) All the uninoculated checks (20 hills) remained healthy. Throughout the growing season of 1925 the plants were under close observation and aside from the fact that transmission was proved beyond a doubt, a number of other facts were obtained which are of interest. Of the three diseased plants in plot (5), two of them represented cuttings from the same mother plant, while the third, although duplicated by another hill, remained the only one showing the disease. Again, the one diseased plant of plot (2) was duplicated by another hill which represented a cutting from the same mother plant. This plant likewise remained healthy in appearance during the entire growing season of 1925. In other words the disease producing agent was either absent or without noticeable effect in some plants, though clearly present in others which were derived from the same mother plant. This it will be recalled was also noted in the 1920 infections. Another striking illustration of the more or less localized infections obtained at times in this disease is the presence of diseased and healthy shoots or runners on the same plant. A clear idea of this phenomenon may be obtained from the following table, representing the plants artificially infected in 1925:

Total Number of Runners	Number of Diseased Runners
10	8
16	1
14	1
15	2
12	1

Symptoms on diseased runners were unmistakably charac-

istic of sweet potato mosaic, yet to leave no doubt of this, cuttings were made from them and grown in the greenhouse. In every case typical mosaic developed in these plants. As further check on this, cuttings were made from the healthy looking runners of the same plants, some of which were taken from the nodes immediately next to the diseased ones. The plants grown from these healthy runners remained healthy up to the end of a 4 months' growth period.

Another observation of some interest reported to the American Phytopathological Society *(16)* was the inequality of disease symptoms on the runners and on the plants propagated from them; and concomitant with this observation, perhaps related to it, was the increase in severity of disease symptoms which was noted as the plants continued to grow. In most cases the first recognizable symptoms were comparatively mild, being in the form of a slight penciling or mottling of a few leaf blades or parts of blades. Marginal irregularities of blades were slight or absent and there was little or no leaf distortion of any kind. But as the season advanced the disease became more severe, resulting finally in extreme chlorotic discolorations, marginal distortions, marked puckerings, and thread-like forms of blades. What is the explanation for this unequal display of symptoms at different times of the growing season? Is it due to variation in ecological factors? Hardly, because diseased plants of known progeny, representing natural infections, were grown alongside of the artificially infected ones and no such variation in symptoms were to be noted on them. The latter had been propagated for several years and the symptoms appeared comparable irrespective of age of plants or weather or soil conditions. In other words, any conditions which are conducive to the growth of sweet potatoes in general, are apparently proper for the development of the disease.

Another explanation that may be offered for this variation in symptoms is that it represents a number of distinct diseases, all of the class of filterable viruses, which may appear singly or in combination with others, and which may or may not be observable, depending upon the environmental conditions which are favorable for the expression of some one particular disease. This explanation is the one that is at present held for the condition frequently noted in Irish potatoes, known as "degeneration diseases". The work of Schultz (17), Schultz and Folsom *(18)* and *(19)*, Goss *(9)*, and others all shows apparently that a number of distinct diseases of the filterable virus type may at times be present in any one plant. If one accepts the parasitic microorganism theory of mosaic etiology, and Miss Eckerson's recent work *(6)* tends to put it on a firm basis, then presumably a number of different species or varieties of microorganisms are involved in the various types of Irish potato mosa-

ic. This explanation may also be considered for the variation in symptoms noted in sweet potato mosaic.

Another explanation is at present preferable to the writer. It is that as the period of incubation is increased, using this term as the period involved for the disease to be noted macroscopically, the disease becomes more marked and of greater severity because the causal agent has had opportunity of increasing in frequency and hence increasing in its power of disease expression. There are a number of reasons why the writer is tenably accepting this rather than the multiple causal-agents theory. As the foregoing work shows, no disease develops in less than 12 or 13 months of vegetative growth, subsequent to the inoculation. Furthermore, it has yet to be proved (and work is being conducted along this line) that the disease is capable of being transmitted unless the inoculated plant has an opportunity of practically continuous or uninterrupted growth for at least 12 months. In other words, up to the present no infections have been obtained in inoculated plants when growth was interrupted in the fall and when the fleshy roots were not propagated until the following spring. But, aside from the possibility that infections may not be obtainable unless a long and continuous period of growth is utilized, the length of time involved in all the transmission cases, irrespective of continuity of growth, is truly remarkable and gives one the impression of a long, drawnout combat in which the host makes a valiant effort to withstand the attacks of the parasite (note the small percentage of infections) and gradually after a long siege succumbs to its ravages. There is also some evidence that the disease may at times lose at least part of its severity so that a diseased plant is able to make a much better growth than its progenitors. Further evidence is awaited prior to the full acceptance of this. It would be well to recall, in the meantime, that in at least one other mosaic disease, that of sugar cane, Edgerton and Taggart *(7)* have reported that the disease after a time has a tendency of becoming less severe in some plants. The length of incubation, the gradual increase in severity of disease, and the gradual decrease in severity noted occasionally, these three factors are believed to indicate that only one disease is involved and that it is probably caused by one agent.

Field evidence of lack of transmissibility under natural conditions has already been fully set forth *(15)*. This in a large measure confirms the results of artificial infections. But in view of the fact that the disease is well scattered over a number of states and further that no transmission has been obtained in less than 12 consecutive months of growth, how is the disease disseminated, and once present in a particular field, what are the possibilities of its spreading? As to the first ques-

tion, this can be answered with some degree of certainty, namely, by the use of diseased roots, or slips derived from diseased roots, and the continued propagation of such plants. The answer to the second question may perhaps be gleaned from the present work. It is as follows: In the northern limits of sweet potato growing, where the growing season is not as long as in more southern regions, there is little or no opportunity for transmission. In the southern regions, particularly in portions of Florida and perhaps in other regions around the Gulf of Mexico, the disease has considerable opportunity of being transmitted to healthy plants. Parts of plants left in the field after harvest may continue to grow in these localities and thus make possible a full incubation period. It is quite probable that the disease had its origin in some southern point from which it has been scattered about through trade in roots or slips. It may be well to bear in mind that the sweet potato, *Ipomoea batatas* Lam., is a native of tropical America and under those conditions it obtains a growing period involving practically the whole year.

See p. 2 for summary.

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